TRANS-ATLANTIC DEBATE

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Debate: Whether venous perforator surgery reduces recurrences

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Superficial venous surgery and perforator vein surgery, specifically, have a long and varied history in the evolution of vascular surgery, especially because venous disease continues to be extremely common. As with other areas of our specialty, perforator vein procedures have progressed from being purely open operations to becoming less invasive procedures. Despite this, there remains much discussion (as well as overt disagreement) about whether perforator vein surgery is actually appropriate and beneficial in the first place. Surgeons have no level I evidence from randomized controlled studies to determine whether perforator vein surgery does or does not reduce the chances of recurrence of superficial venous varicosities, so we must rely on the evidence as it currently is. Perhaps not surprisingly, our two experts have assembled divergent opinions on the role of perforator venous surgery in contemporary practice. (J Vasc Surg 2014;60:796-803.)

PART I: VENOUS PERFORATOR SURGERY IS PROVEN AND DOES REDUCE RECURRENCES

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There are few areas of superficial venous surgery in which opinions are as polarized as that regarding the role of perforator veins and incompetent perforator veins (IPVs) in the treatment of varicose veins. On one side, perforating veins are regarded as "normal," allowing blood refluxing in incompetent superficial venous trunks to re-enter the system, and thus, they should be left alone,[1] regardless of their size or apparent reflux on certain tests. On the other side, IPVs are seen as different from competent perforating veins, allowing significant venous outflow from the deep system into the superficial venous system and causing morphic changes to the local superficial veins (varicosities or telangiectasia) or tissue (edema or fascia cutaneous changes).[2]

The large number of publications on the subject do not currently provide a definitive answer—hence this debate! However, as practicing clinicians, we are not able to postpone the management of patients presenting with varicose veins or other sequelae of superficial venous reflux disease until the case has been proven beyond doubt.

As such, practicing clinicians need to approach this subject in a pragmatic fashion. We need to treat our patients in accordance with our own observations and experience and be guided by what evidence is currently available. The absence of a definitive randomized controlled trial (RCT) does not mean that the science is unproven—merely that the level of evidence is lower than some might like. A great many procedures are performed daily in our hospitals that have the same or even lower levels of evidence to support them. Merely listing the current publications and available research into IPVs and varicose veins is not sufficient to answer this question satisfactorily because we may end up denying our patients the excellent results that have been reported when perforator veins are treated in conjunction with the treatment of truncal venous reflux.[3]

Before launching into the debate proper, we must acknowledge the difficulty in producing a standard definition of what is a significant IPV.

DIAGNOSIS OF AN IPV

Although most clinicians would accept that a perforating vein is a venous communication between the superficial and deep veins in the leg, "perforating" through the deep investing fascia and, hence, the underlying muscle, the question about what constitutes incompetence and what level of reflux in IPVs is significant, is not exact.

For those who believe that bidirectional flow in perforators is abnormal, many use the diameter of the perforator as a marker of incompetence. However, although >3.9 mm in the subfascial portion indicates reflux, one-

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third of IPVs have diameters of <3.9 mm, meaning that we cannot use size alone to diagnose an IPV.[4] Agreements of pathologic reflux times also vary, with times for reflux in IPVs of >350 ms being proposed rather than the more commonly used >500 ms.[5]

THE ASSOCIATION BETWEEN IPVs AND VARICOSE VEINS+PRIMARY AND RECURRENT VARICOSE VEINS

Although the definition of what constitutes an IPV is not exact, many IPVs are clearly refluxing, and so many associations have been identified between clearly refluxing IPVs and varicose veins. There is a clear association between the presence of IPV and some varicose veins,[6,7] with increasing numbers and sizes of IPVs in progressively worsening varicose veins[6] and increased numbers of IPVs found in legs with recurrent varicose veins.[7] These and other studies show the association between varicose veins and IPVs both above-knee and below-knee. To date, there has not been a clear attempt to separate the above-knee and below-knee IPVs into distinct pathophysiologic entities, and so arguments must not be confused by separating them at this time.

None of these studies have been able to show a causative relationship between IPVs and varicose veins, because when the IPVs reflux blood from the deep system, there is almost always a corruption of valves in a local superficial venous trunk. Hence, when reflux is found in an IPV and also in an associated section of truncal vein, there is no clear way of telling which was cause and which was effect.[6]

However, these studies, coupled with clinical observations of the occasional patients who present with varicose veins arising only from IPVs and improve when these have been treated successfully, have led many clinicians, such as myself, to treat IPV when they are identified. So to return to the question posed, is this venous perforator surgery unproven?

IS VENOUS PERFORATOR VEIN SURGERY UNPROVEN?

If we accept that it is the venous reflux in the IPV that signifies venous pathology and distinguishes an IPV from a normal perforating vein, then the success of perforator vein surgery can be measured by the successful closure or prevention of reflux in these veins. To use more global definitions of success, such as patient-reported outcomes, which has become fashionable in venous surgery, hides the effects of treating or failing to treat an IPV by including confounding variables, such as the treatment of truncal reflux or phlebectomy, which may or may not be associated with the IPV in question.

Studies in the past have suggested that treating truncal reflux in the great saphenous vein (GSV) will allow an IPV to shrink and become competent again.[8,9] Our own study, however, showed this was not the case when the IPVs were followed up over a long enough period, suggesting the previous observations had mistaken acute changes for permanent restoration of function.[10] Such acute changes might be explained by temporary occlusion of the IPV by postoperative thrombophlebitis.

Hence, to permanently stop venous reflux in IPVs in patients with varicose veins, the IPV itself needs to be treated. Before 1985, the only way to do this was ligation by open surgery, as in the Linton operation[11] or the Dodd and Cockett procedure,[12] or by blind disruption such as that proposed by Edwards.[13] In 1985, however, Hauer[14] invented subfascial endoscopic perforating vein surgery (SEPS), allowing an endoscope to be placed in the subfascial space and the IPV to be visualized and clipped, with or without subsequent division.[15] Studies on the efficacy of SEPS to stop reflux in IPVs have shown a midterm technical success rate of 78%.[16]

With the advent of catheter-based endovenous procedures, we invented the transluminal occlusion of perforator (TRLOP) technique in 2001, presented it in 2002,[17] and published it in 2004.[18] TRLOP describes the method of percutaneous cannulation of an IPV under ultrasound guidance through a single needle hole, so that any treatment catheter can be passed into it for thermal or nonthermal ablation. The success of TRLOP at 1 and 5 years was the same or better than that reported for SEPS[19,20] and encouraged other authors to "reinvent" and to attempt to rename the TRLOP technique. Since the original descriptions of TRLOP in 2002 and 2004, terms, such as percutaneous ablation of perforators,[21] ultrasound-guided percutaneous ablation,[22] and other descriptive terms or device names have appeared,[23] although none have added anything to the original description of the TRLOP technique as presented in 2002 and 2004.

Nevertheless, whatever a clinician might erroneously call his or her version of the TRLOP technique, the ability to close the IPV to prevent venous reflux in >80% in the long-term has now been proved. As such, we can clearly conclude that to state that "perforator vein surgery is unproven" is clearly wrong. Now we can turn our attention to the second part of the question—that of reduction of recurrences.

PERFORATOR VEIN SURGERY … DOES NOT REDUCE RECURRENCES?

That perforator vein surgery reduces the recurrence of venous leg ulcers is well proven by individual studies[24-26] and also by a meta-analysis of the available literature.[27] Indeed, O'Donnell himself has been involved in such work,"These findings emphasize the importance of ligating all incompetent perforating veins, as *ulcer* healing was never achieved when residual perforating veins were found at follow-up."[28] Although some might try to argue that it is deep vein reflux in such patients rather than the IPVs that is important, O'Donnell et al[29] were able to reassure us that "deep system reflux as measured with duplex scan valve closure times did not correlate with the rate of *ulcer* healing or recurrence," whereas the treatment of IPVs was of clear benefit. Hence, the treatment of IPVs in venous ulceration is proved to reduce ulcer recurrence.

However, when the same venous reflux is found in the same IPV but in a leg with varicose veins rather than leg

ulcers, O'Donnell and others suddenly stand against the treatment of the IPV as a strategy to reduce recurrences of varicose veins! This is a remarkable turnaround, unless they have proven that the venous reflux in IPV in legs with leg ulcers has a completely different pathophysiology from that in legs with varicose veins. Merely having more numerous or larger IPVs in ulceration is not a sufficient difference, because these changes have been shown to be a result of disease progression.[6] Indeed, the fact that IPVs do become more numerous and larger as venous disease progresses should lead one to the conclusion that these IPVs need treatment to help stop such deterioration—the opposite of what the doctors on the opposing side of this debate are proposing.

The failure of being able to detect a hemodynamic change with air plethysmography after perforator surgery[30] has been used to support the nihilistic view of IPV treatment in varicose veins.[23] However plethysmography in the form of photoplethysmography has already been shown to be "a poor method of assessment of venous reflux after SEPS."[31] The failure of air plethysmography to show a hemodynamic effect after perforator treatment merely suggests that it was the wrong test to use or was not sensitive enough to measure the effect. It does not add to the argument about whether individual IPVs lead to recurrent varicose veins if left untreated.

There is currently no RCT evidence to show that the addition of IPV surgery to truncal vein surgery reduces recurrence. Our own RCT[10] failed to show such an effect due to the overwhelming recurrent reflux caused by neovascularization and strip tract revascularization,[32,33] which hid any effect from the IPVs. However, there is overwhelming circumstantial evidence to support this view, with multiple studies showing IPVs are a major cause of recurrent varicose veins after surgery[34-36] and more recently identified as the most common cause of recurrence after endovenous ablation for varicose veins.[37]

CONCLUSIONS

As shown above, venous perforator vein surgery has been proven and has been shown to be effective at stopping venous reflux in IPV. The reduction of recurrent venous ulceration after treatment of IPV has been proven beyond doubt, and so, unless the sceptics can show a different mechanism of action between venous reflux in IPVs in the legs with venous ulceration compared with the venous reflux in IPVs in legs with varicose veins, then these results can be extrapolated to the treatment of varicose veins.

Although treatment of IPVs has not yet been proven to reduce recurrences, the circumstantial evidence is overwhelming. The studies presented here show that IPVs are associated with varicose veins and that as varicose veins worsen, the numbers of and sizes of IPVs increase. Furthermore, recurrent varicose veins are associated with increased numbers of IPVs, suggesting a causative link. Studies looking at the causes of recurrent varicose veins after open surgery regularly confirm IPVs are a major cause of recurrence, and IPVs have been shown to be the major cause of recurrent varicose veins after endovenous surgery.

Until irrefutable evidence has been produced to the satisfaction of all, the onus is on physicians who support the contention under debate to prove that treating IPVs does not reduce recurrences in view of the overwhelming circumstantial evidence available to the contrary.

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PART II: VENOUS PERFORATOR SURGERY IS UNPROVEN AND DOES NOT REDUCE RECURRENCES

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My position in this debate is that treatment of incompetent perforating veins (IPVs) in association with ablation of the great saphenous vein (GSV) for axial reflux does not reduce recurrent varices after surgery (REVAS). This debate focuses on IPV treatment as an indication to prevent REVAS, not as an indication to promote venous ulcer healing or prevent recurrence, where the argument may be quite different. The argument to not treat IPVs peremptorily at the time of GSV surgery, as a method to prevent recurrence after GSV surgery, is based on:

1. IPVs are not the major cause of REVAS.
2. The treatment of GSV reflux alone will concomitantly correct a significant proportion of IPVs.
3. The interruption of IPVs with many techniques is associated with *residual* or "missed IPVs," and the procedure is not permanent or durable, leading to *true* REVAS of the IPVs.
4. Recurrence is frequently related to progression of chronic venous insufficiency, which is not prevented by preemptory IPV ablation at the time of GSV ablation.

OVERVIEW

Perrin et al[1] led a consensus conference in 1998, which brought both definition and classification to the problem of REVAS—much like the CEAP classification did for the larger area of chronic venous insufficiency.[2] REVAS was defined as "the existence of varicose veins in a lower limb previously operated on for varicose veins with or without adjuvant therapies." REVAS was classified by:

1. Topographic sites, such as the thigh;
2. Source of recurrence (the cause of deep venous reflux into the superficial system), such as the perforators in the thigh or calf; and
3. The nature of the sources, whether the recurrence was at the site of previous surgery or at another new site.

REVAS is customarily divided into anatomic recurrence, which is defined by duplex ultrasound imaging and may be asymptomatic; and clinical recurrence, which is associated with symptomatic recurrent varicosities. Finally, whether the patient underwent treatment of REVAS can be viewed as a patient outcome measure.

IPVs ARE NOT THE MAJOR CAUSE OF REVAS

During the last decade, endovenous ablation (EVA) of the GSV or small saphenous veins (SSVs) by laser ablation (EVLA) or radiofrequency ablation (RFA) has become the principle therapy for varicose veins in the United States rather than ligation and stripping (L&S), and as a result, EVA has increased 450-fold during the last decade.[3] EVA has been recommended as the primary procedure for saphenous incompetence by the Society for Vascular Surgery/American Venous Forum Guidelines for varicose veins[4] and by the United Kingdom National Institute for Health and Clinical Excellence Guidelines.[5] Thus, REVAS associated with EVA becomes an important consideration. L& S of the GSV by the short strip technique usually avoids treatment of the below-knee GSV,[6] whereas EVA may access the below-knee GSV and ablate the upper portion of the below-knee GSV.[3]

REVAS has been well studied after L&S. In a multicenter study from eight countries involving 199 patients with REVAS after L&S, Perrin et al[7] showed that the commonest sites of recurrence were the thigh (68%) and the lower leg (85%). By contrast, that study showed the saphenofemoral region (47%) and thigh perforators (30%) were the major sources of REVAS. The lower leg

IPVs were the source of recurrence in 43% of limbs. Unfortunately, no information was given about the number of limbs that had specific treatment of IPVs at the time of the initial L&S.

Using the REVAS classification system, Bush et al[8] reported on REVAS from seven centers treating 2380 patients, of which a strikingly low 164 (7%) developed REVAS at a median of 3 years.[8] EVLA was performed as the initial procedure in 80% of patients, whereas the older RFA catheter was used in most of the RFA procedures. No information was provided on whether IPVs were treated with the original ablation. The authors used the all-encompassing term of "perforators" for the source of reflux. When the specific anatomic site was defined, perforators in the thigh, rather than in the calf, were associated with a statistical increase in GSV recanalization. In their analysis, REVAS appears to be defined as reflux on duplex imaging, and the incidence of clinical REVAS is difficult to tease out. Of interest to this debate, REVAS developed at new previously nonrefluxing sites—16% at the SSV and 24% at the anterior accessory (AA) GSV. Thus, 40% of all REVAS was due to disease progression and not amenable to preemptory treatment of the IPVs at the time of EVA.

The least biased information and of highest evidentiary value about REVAS after EVA can be derived from randomized controlled trials (RCTs), where the data are collected prospectively through a uniform protocol, particularly with duplex ultrasound follow-up and preferably using the REVAS classification.[3] Those RCTs where no specific treatment was provided to the IPVs were examined. In an earlier and smaller RCT, Perala et al[9] described their findings at 3 years after RFA in 15 patients. The cause of REVAS, which occurred in 33% of their patients, was reflux in an AASV or in a patent duplicate GSV. No IPVs were detected.

Rasmussen et al[10] compared EVLA (67 limbs in 60 patients) with L&S (67 limbs in 58 patients) in a RCT over a 5-year period. The varicosities of the patients in this RCT were treated with stab phlebectomies for EVA and L&S, but no specific treatment was directed at IPVs. At 5 years, there was no difference in clinical REVAS between EVLA (47%) and L&S (55%), but retreatment principally by sclerotherapy was required in a lesser proportion (39%). Reflux was found in the AASV in 24% of the limbs and in the thigh perforators in another 20%, but calf IPVs accounted for only 16%. Disselhoff et al[11] compared EVLA vs L&S in 120 patients, and at 2 years, calf IPVs were not described as a cause of REVAS.

THE TREATMENT OF GSV REFLUX ALONE WILL CONCOMITANTLY CORRECT A SIGNIFICANT PROPORTION OF IPVs

Stuart et al[12] were one of the first groups to demonstrate a reduction in the number of duplex-detected IPVs after L&S of the GSV from 65% of limbs preoperatively to 37% postoperatively ($P < .01$), whereas the proportion of IPVs declined from 52% to 28%. They found, however, that deep venous incompetence adversely affected the reduction in IPVs with L&S. Table I reports a similar significant decrease in the proportion of limbs with IPVs after L&S in several other series of predominantly CEAP $C_{2\text{-}3}$ patients.[13,14] The Gohel et al[15] study of duplex follow-up of the Effect of Surgery and Compression on Healing and Recurrence (ESCHAR) RCT, where all patients were $C_{5\text{-}6}$ and many had deep venous incompetence, found a smaller but significant decrease in postoperative IPVs. In these combined series, the average decrease in of IPVs after treatment of the GSV postoperatively was 50%.

Table I. The effect of great saphenous vein (GSV) treatment by ligation and stripping (L&S) on incompetent perforating veins (*IPVs*)

First author	*Year*	*Limbs, No.*	*Exam time, weeks*	*IPVs, No. (%)*		
				Pre-op	*Post-op*	*New*
Stuart[12]	1998	62	14	40 (65)	23 (37)	
Mendes[13]	2003	24	12	24 (100)	8 (33)	
Blomgren[14]	2005	103	8	42 (100)	23 (45)	8 (18)
Gohel[15]	2005	115		59 (52)	44 (43)	12 (12)

THE INTERRUPTION OF IPVs WITH MANY TECHNIQUES IS ASSOCIATED NOT ONLY WITH *RESIDUAL* OR MISSED IPVs, BUT ALSO THIS PROCEDURE IS NOT PERMANENT OR DURABLE, LEADING TO *TRUE* REVAS OF IPVs.

To advocate a procedure that is preventative, the technique must have a high initial success rate and the procedure must be effective long enough to garner the proposed late benefits against REVAS. The current treatment of IPVs has evolved from the open procedures of Linton[16] and Cockett,[17] where all perforating veins were visualized and ligated by using a long medial subfascial incision, through SEPS, where the IPVs were selectively ablated by an endoscopic approach,[18,19] to the current technique of direct percutaneous thermal or sclerotherapy treatment of the IPVs under ultrasound guidance.[20]

Our own experience showed an early ultrasound residual or "missed IPV" rate of 22% in 19 limbs after SEPS,[21] which is similar to the residual rate of 20% described by Sybrandy et al[22] in their 40-patient RCT, which compared SEPS with the Linton procedure (0% residual rate). On follow-up duplex examination, the large 200-limb Dutch SEPS RCT revealed at least one residual/missed IPV in 50% of the procedures.[23] As the former trial suggests, SEPS is highly operator dependent, and this is underscored by the Kolvenbach et al[24] redo SEPS series of 19 patients, which was principally referral-based, presumably from lower-volume centers. Besides technical problems with residual IPVs, progression of a perforator, which was normal on the initial duplex assessment, to an IPV frequently occurs. In the REVAS classification, this has been defined

Table II. Comparison of the occlusion/residual rate with direct percutaneous ablation of perforators to residual incompetent perforating veins (IPVs) after subfascial endoscopic perforator vein surgery (*SEPS*)

First author	*Year*	*Limbs, No.*	*Perfs, No.*	*F/U, months*	*Occluded/ residual, %*
RFA					
Chang[26]	2005		38	6	63/37
Lumsden[27]	2006	55	97	12	44/56
Elias[28]	2007	…	20	0.75	81/19
van den Bos[29]	2009	12	14	0.25	100/0
Bacon[30]	2009	58	125	60	66/34
Hingorani[31]	2009	38	48	1	88/12
Lawrence[32]	2011	51	86	…	58/42[a]
				24	79/29
Laser					
Proebstle[33]	2007	60	67	3	99/1
Kabnick[34]	2006	…	25	4	85/15
Elias[28]	2007	…	50	…	90/10
Murphy[35]	2006	…	100	…	90/10
Sclerotherapy					
Masuda[36]	2006	80		…	98/2[a]
				20	75
Kiguchi[37]	2014	62	189	36	54/46
SEPS					
Iafrati[21]	1997	15	18	5.5	22
Roka[25]	2006	92		0.25	100
Sybrandy[22]	2001	20	…	1.5	28
Linton[16]	2001	19	…	…	0

F/U, Follow-up; *RFA,* radiofrequency ablation.
[a]First row of data indicates the initial findings.

as new REVAS. Sybrandy et al[22] observed that the initial 0% residual IPV rate with the Linton procedure had climbed to a 45% new REVAS rate in later follow-up, whereas after SEPS, this figure had doubled to a 42% new REVAS rate. After SEPS in 92 limbs, no residual IPVs were observed on duplex in the Roka et al[25] series, but new REVAS of IPVs developed in 20 limbs (20%) during a mean follow-up of 3.7 years.

The current techniques of thermal ablation of IPVs, such as transluminal occlusion of perforator veins (TRLOP) or percutaneous ablation of perforators (PAPS), is hampered by considerable operator variability with a steep learning curve.[20] Table II summarizes the incidence of residual IPVs after PAPS in the published literature as well as from series presented at meetings. The rate of IPV occlusion after RFA-PAPS varies from two series at 60% (40% residual REVAS rate)[26,27] to several at ≥90%.[28,29,31] Laser-PAPS appeared to have somewhat better early occlusion rates.[33-35] The appreciable learning curve of PAPS is best appreciated from the large experience of Lawrence et al,[32] where the initial success rate with RFA-PAPS was 58% and rose to 79% after 2 years of experience.

Strong further evidence on the lack of permanence of IPV treatment (true REVAS) is provided by the large prospective study of van Rij et al,[38] who monitored 145 limbs with 850 IPVs, which were directly ligated under duplex ultrasound guidance. To distinguish between residual IPVs and new IPVs causing REVAS, the limbs were topographically mapped at the initial treatment for subsequent serial postoperative duplex examinations. At 3 years, they observed that 76% of limbs had developed 380 further IPVs. Of these, 152 (40%) recurred at the site of the IPV ligation due to neovascularization—same site REVAS; most importantly, 225 (59%) previously normal perforators by duplex examination increased their diameter and became incompetent over the follow-up period—new REVAS, which is indicative of disease progression. The sole series of direct thermal ablation of IPVs with long-term data comes from my debate opponent's unit.[30] The authors described the results of an audit of 82 of the 106 initially treated patients (25 were excluded due to distance). Sixty-seven patients responded, of whom 37 agreed to participate (35% of the original cohort and 55% of the respondents). Of the 125 IPVs originally treated, 81% were closed (21% open) in 20 limbs (34% of limbs), and 24% of the limbs demonstrated new IPVs.

Kiguchi et al[37] and associates treated 62 C_6 patients with repeated sclerotherapy under duplex guidance and found a low 54% had occluded at a mean of 30.2 months. Finally, the case series by Masuda et al[36] illustrates the recurrence of incompetence in IPVs after ultrasound-guided liquid sclerotherapy of IPVs in 80 limbs ($C_{2\text{-}4}$ in 70%). Although 98% of the IPVs were occluded initially on duplex ultrasound, only 75% remain occluded at 1½ years.

RECURRENCE OVER TIME IS RELATED TO PROGRESSION OF CHRONIC VENOUS INSUFFICIENCY

Disease progression has been defined as a result of the natural history and evolution of the disease, where the involved varicosities are not dilated and varicose at the time of the initial treatment but develop reflux due to the "natural history of the disease process."[39] Van Rij et al[40] monitored 92 patients with 127 limbs that underwent L&S of the GSV with concomitant duplex ultrasound-guided direct ligation of significant IPVs. Serial duplex and air plethysmography studies of these patients showed 13.7% had clinical evidence of recurrence at 3 months, and this figure jumped to 51.7% at 3 years. The venous filling index, a measure of reflux, progressively increased in a great proportion of limbs during the follow-up period. This indicated a physiologic recurrence that paralleled and preceded clinical recurrence. Despite ligation of all significant IPVs at the initial surgery, new IPVs rose to 59% and 90% of limbs at 2 and 3 years, respectively, which is indicative of the major role of disease progression in REVAS. The previously quoted Recurrent Veins After Thermal Ablation (REVATA) study of Bush et al[8] found that that 40% of all REVAS was due to disease progression in new sites—16% at the SSV and 24% at the AAGSV—that were all previously normal.[8]

CONCLUSIONS

One can only conclude that preemptive treatment of IPVs at the time of GSV surgery is not associated with prevention of recurrence. The best strategy for treatment of recurrent varicose veins after GSV ablation and removal of varicosities may be similar to that used for arterial occlusive disease or dental carries, with periodic check-ups and treatment as the problem arises.

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EDITORS' COMMENTARY

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In the midst of this spirited discussion there is one issue where our debaters agree, namely the role of incompetent perforator vein (IPV) interruption in promoting venous ulcer healing or preventing recurrence. This is consistent with the Practice Guidelines of the Society for Vascular Surgery and the American Venous Forum that recommends "treatment of pathologic perforating veins that includes those with outward flow ≥500-ms duration, with a diameter ≥3.5 mm, located beneath healed or open venous ulcer (class C_5-C_6)."[1] This same document, however, does not recommend treatment of IPVs in patients with simple varicose veins (class C_2), based on a "moderate" level of evidence. This area of contention has prompted this discussion by our experts and it is evident that there are several areas of disagreement.

In his argument in favor of interruption of IPVs to reduce varicose vein recurrence, Prof Whitely outlines the literature describing an association between IPVs and varicose veins but readily admits that a causal relationship has not been definitely proved, as is the case with venous ulcerations. He proposes a common pathophysiology and shared role of IPVs between venous ulcers and varicose veins that, he argues, would validate IPV surgery with varicose veins as it does with venous ulcers. Interruption of IPVs is possible with a high degree of success (>80%) with increasingly less invasive techniques including those pioneered by Professor Whitely, but is it necessary? He argues that it is necessary and is supported by "overwhelming circumstantial evidence."

Dr O'Donnell counters with the argument that venous stasis ulcers and varicose veins do not share a common pathophysiology and that IPVs are not the major cause of recurrent varicosities, which are a result of the natural history of the disease itself, irrespective of IPV status. Regardless, he argues, IPV surgery is not as successful as its proponents claim, with missed veins and less-than-optimal durability.

This leaves us without a definitive answer. The role for perforator vein surgery in advanced venous disease, or venous ulcers, seems clear, but remains less so with lesser degrees of disease or varicose veins. Although IPV surgery can be done with some success by less invasive techniques, the question remains about whether it has any value in decreasing the risk of recurrent varicosities. The current level of evidence does not support its routine use in C_2 disease and we should await further evidence before recommending its wider adoption.

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